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Impact of hospital volume and type on outcomes of open and endovascular repair of descending thoracic aneurysms in the United States Medicare population

Virendra I. Patel, MD, MPH, Shankha Mukhopadhyay, MS, Emel Ergul, MS, Nathan Aranson, MD, Mark F. Conrad, MD, Glenn M. LaMuraglia, MD, Christopher J. Kwolek, MD, and Richard P. Cambria, MD, *Boston, Mass*

Objective: Favorable outcomes of thoracic endovascular aortic repair (TEVAR) compared with open repair for descending thoracic aortic aneurysms (DTAs) have led to increasing TEVAR use. We evaluated the effect of case volume and hospital teaching status on clinical outcomes of intact DTA repair.

Methods: The Medicare Provider Analysis and Review (MEDPAR) data set (2004 to 2007) was queried to identify open repair or TEVAR for DTA. Hospitals were stratified by DTA volume into high volume (HV; ≥ 8 cases/y) or low volume (LV; < 8 cases/y) and teaching or nonteaching. The effect of hospital variables on the primary study end point of 30-day mortality and secondary end points of 30-day complications and long-term survival after open repair and TEVAR DTA repair were studied using univariate testing, multivariable regression modeling, Kaplan-Meier survival analysis, and Cox proportional hazards regression modeling.

Results: We identified 763 hospitals performing 3554 open repairs and 3517 TEVARs. Overall DTA repair increased ($P < .01$) from 1375 in 2004 to 1987 in 2007. The proportion of hospitals performing open repair significantly decreased from 95% in 2004 to 57% in 2007 ($P < .01$), whereas those performing TEVAR increased ($P < .01$) from 24% to 76%. Overall repair type shifted from open (74% in 2004, the year before initial commercial availability of TEVAR) to TEVAR (39% open in 2007; $P < .01$). The fraction of open repairs at LV hospitals decreased from 56% in 2004 to 44% in 2007 ($P < .01$), whereas TEVAR increased from 24% in 2004 to 51% in 2007 ($P < .01$). Overall mortality during the study interval for open repair was 15% at LV hospitals vs 11% at HV hospitals ($P < .01$), whereas TEVAR mortality was similar, at 3.9% in LV vs 5.5% in HV hospitals ($P = .43$). LV was independently associated with increased mortality after open repair (odds ratio, 1.4; 95% confidence interval, 1.1–1.8; $P < .01$) but not after TEVAR. There was no independent effect of hospital teaching status on mortality or complications after open repair or TEVAR repair.

Conclusions: The total number of DTA repairs has significantly increased. Operative mortality for TEVAR is independent of hospital volume and type, whereas mortality after open surgery is lower at HV hospitals, suggesting that TEVAR can be safely performed across a spectrum of hospitals, whereas open surgery should be performed only at HV hospitals. (J Vasc Surg 2013;58:346–54.)

The feasibility of thoracic endovascular aortic repair (TEVAR) for descending thoracic aortic aneurysms (DTAs) was first reported by Dake et al¹ in 1994. Subsequent industry-sponsored comparative trials have shown that TEVAR is associated with reduced perioperative morbidity and mortality compared with open DTA repair.^{2–5} Results from these device trials led to United States Food and Drug Administration (FDA) approval for the first commercial use of TEVAR for DTA repair in 2005.

In addition, the operative mortality and morbidity benefits of TEVAR from single-center reports^{6–8} and device registries^{9,10} have further supported the findings from the comparative trials.^{2–4} This has led to widespread acceptance of TEVAR for the management of DTA in contemporary practice. As such, in the years after FDA approval, TEVAR has supplanted open surgical repair in most patients with an anatomically suitable DTA.^{11–13}

Previous publications have only reported the effect of hospital volume on outcomes of open DTA repair,¹⁴ without addressing the effect of hospital teaching status on outcomes. Despite the widespread use of TEVAR, the effects of hospital type and volume on outcomes of DTA repair in the age of TEVAR are as yet unknown. The goal of this study was to evaluate the effects of hospital teaching status and hospital volume on operative mortality and outcomes after DTA repair.

METHODS

Data set. This study evaluated the effects of hospital type and procedural volume on outcomes of DTA repair using

From the Division of Vascular and Endovascular Surgery, Massachusetts General Hospital, Harvard Medical School.

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Reprint requests: Virendra I. Patel, MD, Massachusetts General Hospital, 15 Parkman St, Wang ACC 440, Boston, MA 02114 (e-mail: vpatel4@partners.org).

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the Medicare Provider Analysis and Review (MEDPAR) data set from 2004 to 2007. The MEDPAR file contains Part A claims data for all Medicare admissions and includes demographic information, admission International Classification of Diseases, 9th Revision (ICD-9) codes, procedural ICD-9 codes, discharge data, and hospital cost data. Linkage with the Medicare Denominator and Vital Statistics files provided geographic and survival information for the study cohort. The MEDPAR data set was queried to identify all patients with an ICD-9 diagnosis of intact DTA (441.2) and a procedure code for open repair (38.35 or 38.45—replacement of thoracic vessel with repair/anastomosis) or TEVAR repair (39.73—endovascular implant graft thoracic aorta; 39.79 was used for years before 39.73 was available). The study excluded patients with ruptured thoracic aneurysms (441.1, 441.5), thoracoabdominal aneurysms (441.6, ruptured; 441.7, intact), thoracic aortic dissection (441), those with a diagnosis of ascending aortic aneurysms, those undergoing concomitant coronary or cardiac valve procedures, and those treated with cardioplegia. Patients were categorized by ICD-9 diagnosis as having undergone thoracic aortic replacement or repair, without anatomic distinction for arch or DTA disease. Given the use of hypothermic circulatory arrest (HCA) for all aortic arch reconstruction and its very limited use for DTA repair at some centers, patients undergoing HCA were excluded.

Study design. This retrospective study evaluated the effect of hospital teaching status (teaching vs nonteaching) and hospital volume (high volume [HV] vs low volume [LV]) on the primary outcome measure of 30-day mortality in patients undergoing open or TEVAR repair of DTA. Secondary outcome measures included any complication and long-term survival. Hospital teaching status was as defined by the MEDPAR data set (yes or no).

Hospitals performing TEVAR and open DTA were stratified as HV or LV according to annual volume. Those hospitals performing >50th percentile of all DTA procedures were defined as HV hospitals. Annual case volume at HV hospitals exceeded eight DTA procedures per year (mean, $22 \pm 16/y$), whereas LV hospitals performed eight or fewer procedures annually (mean, $2 \pm 1/y$). Additional thresholds to define HV were evaluated without affecting study conclusions; therefore, the 50th percentile was chosen for final analysis.

Comorbidities were identified using ICD-9 codes for diabetes mellitus (250.0-250.9), hypertension (401.0-405.9), chronic obstructive pulmonary disease (490.0-496.0), ischemic heart disease (410.0-414.9), cerebrovascular disease (430.0-438.0), and peripheral arterial disease (440.0-440.9, 443.0-443.9). Complications were similarly identified using ICD-9 codes.

Data analysis. Categorical variables are presented as absolute number and percentages, with continuous data presented as mean values \pm standard deviation. Univariate analysis was performed by using the χ^2 or the Fisher exact test for discrete variables, the *t*-test with equal variances

for normally distributed continuous variables, and the Wilcoxon rank-sum test for non-normally distributed continuous and ordinal variables. Analyses for trends across time were performed using a χ^2 trend test. Multivariable regression analyses were performed to identify independent predictors of perioperative (30-day) death or any complication. Kaplan-Meier analysis was used to assess actuarial survival, with differences between the areas under the curves determined by the log-rank test. Proportional hazards regression analysis was performed to evaluate for risk-adjusted effect of hospital type on late survival. $P < .05$ was considered significant. All statistical analysis was performed using SAS 9.2 software (SAS Institute, Cary, NC).

RESULTS

Our study consisted of 7071 patients, 3554 open and 3517 TEVAR, who underwent DTA repair at 763 hospitals during the study period. The total annual number of DTA repairs significantly increased, from 1375 in 2004 to 1987 in 2007 ($P < .001$), driven mainly by a significant increase in TEVAR with a significant decrease in open repair (Table I). By 2007, the proportion of open procedures had decreased to 38% compared with 74% in 2004. A significant increase occurred in the number of hospitals, teaching hospitals, nonteaching hospitals, and LV hospitals performing DTA repair over time (Table II), whereas the number of HV hospitals remained stable. The proportion of hospitals performing open DTA repair significantly decreased, but the number of hospitals performing TEVAR significantly increased (Table II). This trend was experienced by all hospital types, which increasingly adopted TEVAR over open repair.

The proportions of open DTA repairs performed at teaching and nonteaching hospitals remained stable during the study period, whereas open cases shifted from LV to HV hospitals (Table III, A). Similarly, the proportions of TEVAR cases performed at teaching and nonteaching hospitals remained stable (Table III, B). TEVAR cases shifted from HV hospitals to LV hospitals during the study period, with a sevenfold increase in the number of TEVARs performed at LV hospitals, from 84 in 2004 to 618 in 2007 (Table III, B).

Demographic and clinical features of patients undergoing open or TEVAR repair stratified by hospital teaching status and volume are presented in Table IV. Univariate analysis shows that patients treated at teaching hospitals were younger, independent of repair type, and those undergoing TEVAR were less likely to be white, have coronary artery disease (CAD), and peripheral vascular disease (PVD) than at nonteaching hospitals (Table IV, B). Patients undergoing open repair at HV centers were more likely to be white and have CAD, PVD, and cerebrovascular disease (Table IV, A) than those treated at LV hospitals. The rest of the clinical and demographic features were similar across hospital type and volume strata (Table IV) by univariate analyses. HV hospitals were more likely to be teaching hospitals (93% HV vs 66% LV; $P < .001$).

Table I. Variation in descending thoracic aneurysm repair procedures over time^a

Procedure	Total, No.	Year			
		2004, No.	2005, No.	2006, No.	2007, No.
All	7071	1375	1697	2013	1987
Open	3554	1024	924	817	746
TEVAR	3517	351	773	1196	1231

TEVAR, Thoracic endovascular aortic repair.

^a $P < .001$ for all variables.**Table II.** Proportions of hospitals performing descending thoracic aneurysm repair over time

Hospital type	Total	Year					P
		2004	2005	2006	2007		
All hospitals, No.	763	359	382	475	496	<.0001	
Open, %		95	82	62	57	<.0001	
TEVAR, %		24	50	73	76	<.0001	
Teaching, No.	510	270	284	348	357	<.0001	
Open, %		95	85	63	61	<.0001	
TEVAR, %		28	52	78	79	<.0001	
Nonteaching, No.	253	89	98	127	138	<.0001	
Open, %		97	71	60	48	<.0001	
TEVAR, %		10	44	61	68	<.0001	
Low-volume, No.	723	319	343	436	456	<.0001	
Open, %		95	80	59	54	<.0001	
TEVAR, %		17	44	71	74	<.0001	
High-volume, No.	40	40	39	39	40	.38	
Open, %		100	100	97	97	.048	
TEVAR, %		75	97	100	100	<.0001	

TEVAR, Thoracic endovascular aortic repair.

Univariate outcomes of open and TEVAR procedures stratified by hospital type are presented in Table V. Despite baseline clinical and demographic differences (as shown in Table IV), risk-adjusted (age, sex, race, hypertension, diabetes, CAD, PVD, chronic kidney disease, hospital volume, and teaching status) multivariable modeling showed no independent effect of teaching status on operative mortality for open (odds ratio [OR], 1.28; 95% confidence interval [CI], 0.9-1.8; $P = .16$) or TEVAR (OR, 1.24; 95% CI: 0.8-1.9; $P = .37$) repair. Multivariable analysis also showed that operative mortality was reduced when open surgery (OR, 0.72; 95% CI, 0.6-0.9; $P < .01$) was performed at HV hospitals; however, operative mortality after TEVAR at HV hospitals (OR, 0.9; 95% CI, 0.1-1.3; $P = .76$) was similar to that at LV hospitals. Risk-adjusted complication rates were similar for open (OR, 1.1; 95% CI, 0.9-1.4; $P = .3$) and TEVAR (OR, 1.3; 95% CI, 0.9-1.6; $P = .06$) when performed at teaching hospitals. The risk of complications after open repair at HV hospitals (OR, 1.1; 95% CI, 0.9-1.3; $P = .24$) was similar to that at LV hospitals. Complications after TEVAR were higher at HV hospitals (OR, 1.5; 95% CI, 1.3-1.8; $P < .01$). Multivariable

predictors of increased 30-day mortality and complications are presented in Tables VI, A and B, respectively.

The long-term survival of patients undergoing open or TEVAR repair was similar (log-rank, $P > .05$) at teaching and nonteaching hospitals (Table VII). The long-term survival of patients undergoing TEVAR was similar (log-rank $P = .7$) at LV and HV hospitals (Table VII), whereas the long-term survival of patients undergoing open repair (Fig) was higher (log-rank, $P < .05$) at HV hospitals. Proportional hazards regression showed that HV hospitals were associated with improved risk-adjusted (baseline clinical features) long-term survival (hazard ratio [HR], 0.82; 95% CI, 0.73-0.92; $P < .01$) after open surgery, whereas teaching hospitals were associated with lower long-term survival (HR, 1.3; 95% CI, 1.1-1.6; $P = .01$). Patients undergoing TEVAR had similar long-term survival hazards independent of hospital volume (HR 1.03 for HV; 95% CI, 0.91-1.2; $P = .6$) or teaching status (HR 0.9 for teaching; 95% CI, 0.783-1.1; $P = .2$). Multivariable predictors of increased late mortality are presented in Table VIII.

DISCUSSION

Since FDA approval of the first commercially available thoracic stent graft in 2005, there has been a significant increase in the use of TEVAR for repair of DTA. This has resulted in expanding indications¹¹ and application of TEVAR to higher-risk patients for whom open surgery was previously considered prohibitive.¹⁵⁻¹⁷ Our previous work¹² showed that in ≤ 3 years (2005 to 2007) of FDA approval, there was a significant expansion in TEVAR use and increasing application of TEVAR for acute aortic pathologies, all of which were off-label use.

This report furthers that effort and shows that there has been not only a rapid expansion in TEVAR use but also an increasing adoption of TEVAR across all hospital types independent of teaching status or hospital volume. Most dramatic has been the consistent annual increase in TEVAR procedures at LV hospitals reflecting an increasing number of new hospitals incorporating DTA repair with TEVAR into their scope of practice. Increased commercial accessibility of TEVAR devices, relative ease of use and deployment, and expanding endovascular experience has likely contributed to this expansion.

Concomitantly, the absolute numbers and proportions of hospitals performing open DTA repair have decreased independent of hospital volume and type. This likely relates to the significant resource utilization by health care systems, the significant physiologic and recovery impact to patients, and the technical challenges required of surgeons involved in open DTA repair. The small annual decreases in open procedures at HV hospitals coupled with the consistent increases in TEVAR at all hospital types and the increasing number of annual DTA repairs suggests an increasing application of TEVAR to an expanding population of patients, without significant increases in referral to HV centers, even for open surgery.

The proportion of open procedures performed at HV hospitals has steadily increased, whereas the absolute

Table III. A, Proportions of open descending thoracic aneurysm repairs by hospital type

Hospital type	Year					P
	2004-2007 (n = 3554), %	2004 (n = 1023), %	2005 (n = 924), %	2006 (n = 833), %	2007 (n = 774), %	
Teaching	89	88	89	89	89	.86
Nonteaching	11	12	11	11	11	.86
Low-volume	49	56	49	46	44	<.001
High-volume	51	44	51	54	56	<.001

Table III. B, Proportions of endovascular descending thoracic aneurysms repair by hospital type

Hospital type	Year					P
	2004-2007 (n = 3554), %	2004 (n = 1023), %	2005 (n = 924), %	2006 (n = 833), %	2007 (n = 774), %	
Teaching	85	87	87	85	84	.31
Nonteaching	15	13	13	15	16	.31
Low-volume	46	24	40	51	51	<.001
High-volume	54	76	60	49	49	<.001

Table IV. A, Clinical and demographics features of patients undergoing open descending thoracic aneurysm repair by hospital type

Variable ^a	All patients (N = 3554)	Teaching (n = 3161)	Nonteaching (n = 393)	P	Low-volume (n = 1782)	High-volume (n = 1772)	P
Age, years	72 ± 8.0	73 ± 8.1	74 ± 7.7	.002	72 ± 7.9	72 ± 8.1	.75
Sex				.93			.27
Male	50	50.1	50		49	51	
Female	50	49.9	50		51	49	
Race				.25			.018
White	88	87	89		86	89	
Other	12	13	11		14	11	
Comorbidities							
HTN	59	58.4	62	.23	57	60	.096
DM	8.3	8.2	9.2	.53	9	7.7	.16
CAD	26	26	25	.51	24	28	.003
COPD	36	36	36	.81	35	37	.23
CVD	5	4.9	5.3	.72	5.5	4.5	.15
PVD	12	12	7.9	.014	7.6	16	<.001
CKD	4.8	5.2	1.5	.001	3.4	6.2	.0001

CAD, Coronary artery disease; CKD, chronic kidney disease; COPD, chronic obstructive pulmonary disease; CVD, cerebrovascular disease; DM, diabetes mellitus; HTN, hypertension; PVD, peripheral vascular disease.

^aContinuous data are shown as mean ± standard deviation and categorical data as percentage.

numbers of open procedures performed at HV centers has slightly decreased. This suggests some degree of regionalization as LV hospitals abandon open surgery in favor of TEVAR; however, the number of actual open DTA repairs performed at LV hospitals remains substantial (~700 cases in 2006 and 2007), which has mortality and survival implications.

Several publications have previously defined the importance of hospital volume on outcomes of complex aortic procedures. Cowan et al,¹⁸ using the National Inpatient Sample (NIS), noted that high hospital volume was an independent predictor of lower hospital mortality

compared with medium-volume or LV hospitals performing thoracoabdominal aneurysm repair. More recently, using the same database, Schermerhorn et al¹⁴ similarly noted the importance of hospital volume on outcomes of open DTA repair. In this series (1988 to 2003), predating commercial availability of TEVAR, LV (1 DTA repair/y) or medium-volume (2 to 3/y) hospitals were independently associated with increased hospital mortality (OR, 1.3; 95% CI, 1.1-1.7; *P* < .05) for open DTA repair.¹⁴

Unlike these reports, which defined volume as low, medium, or high according to volume terciles, we defined hospital volume as HV or LV by the 50th percentile in

Table IV. B, Clinical and demographic features of patients undergoing endovascular thoracic aortic repair by hospital type

Variable ^a	All patients (N = 3517)	Teaching (n = 3001)	Nonteaching (n = 516)	P	Low-volume (n = 1759)	High-volume (n = 1758)	P
Age, years	75 ± 7.8	75 ± 7.9	76 ± 7.3	.018	75 ± 7.7	75 ± 7.9	.94
Sex				.82			.35
Male	58	58	58		57	59	
Female	42	42	42		43	42	
Race				<.0001			.84
White	86	85	92		86	86	
Other	14	15	7.8		14	14	
Comorbidities							
HTN	70	71	68	.14	72	69	.053
DM	13	13	11	.11	13	13	.81
CAD	34	33	41	.0005	35	33	.24
COPD	41	41	42	.72	40	42	.36
CVD	4.5	4.5	4.6	.88	4.6	4.4	.81
PVD	16	15	19	.023	16	15	.86
CKD	9.9	10	9.1	.52	10	9.5	.43

CAD, Coronary artery disease; CKD, chronic kidney disease; COPD, chronic obstructive pulmonary disease; CVD, cerebrovascular disease; DM, diabetes mellitus; HTN, hypertension; PVD, peripheral vascular disease.

^aContinuous data are shown as mean ± standard deviation and categoric data as percentage.

Table V. A, Clinical outcomes after open aortic repair by hospital type

Variable	All hospitals (N = 3554), %	Teaching (n = 3161), %	Nonteaching (n = 393), %	P	Low-volume (n = 1782), %	High-volume (n = 1772), %	P
30-day death	12	12	12	.94	15	11	.0048
Any complication	50	50	46	.084	47	51	.044
Bleeding	17	17	14	.19	16	17	.5
Cardiac	13	13	10	.14	12	13	.54
Infectious	7.1	7.4	4.8	.065	5.9	7.5	.12
Pulmonary	23	23	19	.078	21	23	.17
Graft	1.5	1.5	1	.41	1	1.6	.21
Renal failure	19	19	16	.063	17	20	.18

Table V. B, Clinical outcomes after endovascular aortic repair by hospital type

Variable	All hospitals (N = 3517), %	Teaching (n = 3001), %	Nonteaching (n = 516), %	P	Low-volume (n = 1759), %	High-volume (n = 1758), %	P
30-day death	5.2	5.3	4.5	.42	3.9	5.5	.09
Any complication	30	30	25	.0094	24	31	.0004
Bleeding	13	13	11	.076	11	13	.19
Cardiac	4.9	5.1	3.9	.25	3.2	5.3	.019
Infectious	3.6	3.6	3.7	.95	2.9	3.8	.24
Pulmonary	7.4	7.5	6.8	.55	6.1	7.7	.13
Graft	4.8	5	3.5	.14	3	5.2	.016
Renal failure	6.6	6.7	6.2	.69	5.3	6.9	.14

volume of all DTA repair procedures, in keeping with other investigators.^{19,20} Despite differences in methodology, our threshold for HV (>8 procedures/y) was similar to the Cowan et al¹⁸ report (median 7 procedures/y). In addition, our conclusions regarding the effect of hospital volume are also similar, because open DTA repair at HV hospitals was associated with lower risk-adjusted operative

mortality (OR, 0.56; 95% CI, 0.4-0.7; $P < .01$). Our study has the additional advantage of perioperative mortality data beyond the initial hospitalization and long-term survival analysis, both of which are a limitation of the NIS data set.

Our results showing improved long-term survival in patients undergoing open DTA repair at HV hospitals likely reflect the perioperative mortality benefit afforded

Table VI. A, Multivariable predictors of increased 30-day mortality

Variable	OR (95 % CI)	P
Open repair		
Age, per year	1.04 (1.02-1.05)	<.01
Female sex	1.3 (1.06-1.6)	.01
Low volume	1.4 (1.1-1.8)	<.01
Nonwhite race	1.6 (1.1-2.1)	<.01
Chronic kidney disease	1.8 (1.2-2.8)	<.01
Cerebrovascular disease	2.7 (1.9-3.9)	<.01
TEVAR		
Cerebrovascular disease	3 (1.8-4.9)	<.01

CI, Confidence interval; OR, odds ratio; TEVAR, thoracic endovascular aortic repair.

Table VI. B, Multivariable predictors of increased 30-day complications

Variable	OR (95 % CI)	P
Open repair		
Age, per year	1.02 (1.01-1.03)	<.01
Female sex	1.2 (1.02-1.4)	.01
Nonwhite race	1.4 (1.2-1.8)	<.01
Chronic kidney disease	1.6 (1.1-2.4)	<.01
TEVAR		
Age, per year	1.02 (1.01-1.03)	<.01
High volume	1.5 (1.3-1.8)	<.01
Female gender	1.5 (1.3-1.7)	.01
Chronic kidney disease	1.8 (1.4-2.3)	<.01

CI, Confidence interval; OR, odds ratio; TEVAR, thoracic endovascular aortic repair.

by HV centers because the actuarial survival curves do not diverge or converge long term. Although not specifically studied, this implies open surgical repair in similar-risk patients at LV and HV centers, and therefore, a health care systems-based (operative and postoperative) advantage at HV centers. The importance of the long-lasting effect of hospital volume on patient survival has additional relevance.

Goodney et al²¹ recently reported that patients undergoing open DTA repair had improved long-term survival compared with patients undergoing TEVAR, with loss of perioperative survival advantage of TEVAR by 1 year (87% open vs 82% TEVAR; log-rank, $P = .001$). Furthermore, this advantage of open repair was more pronounced in risk-adjusted and propensity-matched cohorts. Our study did not formally evaluate late outcomes comparing open and endovascular management of thoracic aneurysms; however, similar to Goodney et al,²¹ we noted an early (1-year) advantage of TEVAR at teaching (81% vs 77%) and LV (81% vs 75%) hospitals (Table VII), which was lost at later time points because patients undergoing open repair exhibited better 5-year survivals at all hospital types (Table VII). As such, good-risk patients should be referred for open surgical repair to HV centers for the best early and late patient survival.

TEVAR had been associated with lower perioperative mortality and complication risk, despite application to older and higher-risk patients in the years after FDA approval. Gopaldas et al,¹³ using NIS data, reported perioperative mortality of patients undergoing TEVAR similar to that of patients undergoing open repair, despite age differences of almost 10 years and higher Deyo comorbidity scores in patients treated with TEVAR in the 2 years after FDA approval. Similarly, Walker et al¹⁶ and Bhamidipati et al¹⁷ showed similar increases in TEVAR use in higher-risk populations within the first 2 years after commercial availability of stent grafts, with similar or improved risk-adjusted mortality in patients treated with TEVAR. Our study is the first to show that most of these increases in TEVAR volume have been the early and rapid adoption of TEVAR by LV hospitals.

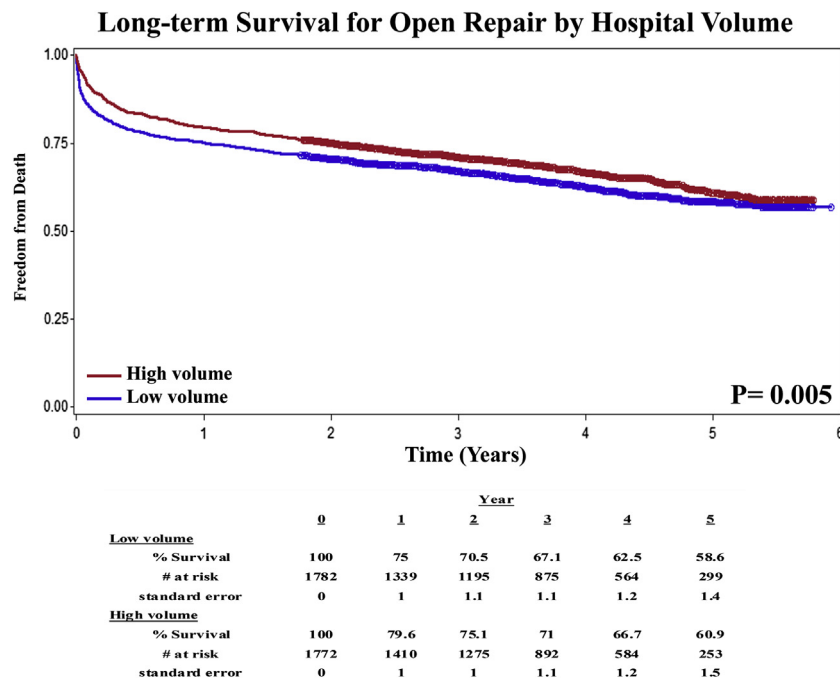
The combination of these data suggests application of TEVAR to higher-risk patients at hospitals with limited experience in the management of patients with DTA. Despite this observation, the annual operative mortality rates of TEVAR at LV and HV hospitals have remained stable over time. In addition, the effect of hospital volume seen with open repair is not observed at hospitals performing TEVAR. Our sensitivity analyses suggested that defining HV on the basis of four TEVAR procedures per year would yield similar conclusions. Our data do show an increased independent risk of any complication (OR, 1.5; 95% CI, 1.3-1.8; $P < .01$), driven mainly by cardiac and graft complications (Table V), after TEVAR performed at HV hospitals. Risk-adjusted analysis using all measured and available factors suggests that other unmeasured yet important factors are contributing to increased perioperative complications at HV hospitals. Long-term survival data, as reported here, suggest no lasting effect of the increased perioperative complications observed at HV hospitals because our findings show similar long-term survival independent of hospital volume.

Our findings show that teaching status defined as yes or no by the MEDPAR data set has no independent effect on perioperative mortality or complications after TEVAR or open DTA repair. Our study results did show an improved long-term survival for patients undergoing open DTA (Fig) at nonteaching hospitals by Cox regression. This may reflect careful patient selection or the effect of unmeasured factors, such as aneurysm anatomy or clinical presentation at nonteaching hospitals, which are not available in our data set. Our findings may also reflect the effect of a systematic error, namely selection bias, because ~70% of hospitals were designated as teaching hospitals and >90% of DTA repairs are performed at teaching hospitals.

In addition, it is unclear whether a hospital's teaching status actually reflects the presence of a surgical training program (with or without vascular fellowship) or a medical training program, or both, which may significantly affect the interpretation of our results. Previous reports have shown that teaching hospitals were associated with reduced mortality and complication rates after complex surgical procedures (eg, pancreatectomy, hepatectomy); however,

Table VII. Survival after open repair or thoracic endovascular aortic repair (TEVAR) by hospital type

Survival	Teaching, %	Nonteaching, %	P	Low-volume, %	High-volume, %	P
Open repair						
1-year	77 ± 1	79 ± 2	.06	75 ± 1	80 ± 1	.0046
3-year	69 ± 1	72 ± 2		67 ± 1	71 ± 1	
5-year	59 ± 1	66 ± 3		59 ± 1	61 ± 1	
TEVAR						
1-year	81 ± 1	80 ± 1	.09	81 ± 1	81 ± 1	.71
3-year	69 ± 1	65 ± 2		68 ± 1	68 ± 1	
5-year	55 ± 2	54 ± 3		58 ± 3	54 ± 2	

**Fig.** Kaplan-Meier survival curves are shown for patients after open descending thoracic aneurysm repair at low-volume and high-volume hospitals.

teaching status was not important compared with hospital volume.²² Our data suggest that this is also the case for DTA repair. Although 93% of HV hospitals were also teaching hospitals, HV hospitals represent only 10% of all teaching hospitals, and regression modeling noted no significant interaction between teaching status and hospital volume. Definitive conclusions regarding the effect of teaching status cannot be ascertained from our study given these limitations; therefore, further study is warranted.

Major limitations of our study include those inherent to large administrative data sets and data availability. Included data are unaudited and based on hospital discharge and procedural billing claims. Whether registered secondary diagnoses represent preoperative conditions or postoperative complications is unclear, and those not likely to positively affect reimbursement might not be included in discharge claims. The specifics of postoperative complications are ill defined; for instance, although ICD-9 codes

are used to report graft complications, the specifics of the severity of the graft complication cannot be known. Similarly, the ICD-9 code for renal complications may include acute tubular necrosis, a temporary increase in creatinine, or dialysis-dependent renal failure. Reported complications therefore represent the spectrum of all complications under a given subheading, and the distribution of more severe or clinically relevant complications is unknown from these data.

Notably absent in administrative data sets are clinical data, such as clinical presentation, operative urgency, aneurysm anatomy, procedural technical details (ie, anesthesia type, estimated blood loss, clamp location, visceral ischemia time, hemodynamic status, length of DTA coverage, compliance with instructions for use, staged debranching, and iliac conduit use), protective adjunct use (ie, cerebrospinal fluid drainage, distal aortic perfusion, and neuromonitoring), and other data that have early and late clinical

Table VIII. Multivariable predictors of increased late mortality after open repair or thoracic endovascular aortic repair (TEVAR)

Variable	HR (95 % CI)	P
Open repair		
Age, per year	1.04 (1.03-1.05)	<.01
Nonwhite race	1.2 (1.01-1.4)	.03
COPD	1.3 (1.1-1.4)	<.01
Low-volume	1.2 (1.1-1.4)	<.01
Teaching hospital	1.3 (1.1-1.6)	.01
Cerebrovascular disease	1.7 (1.4-2.1)	<.01
Chronic kidney disease	1.7 (1.3-2.1)	<.01
TEVAR		
Age, per year	1.03 (1.02-1.04)	<.01
COPD	1.2 (1.1-1.3)	<.01
Cerebrovascular disease	1.5 (1.9-2.0)	.01
Chronic kidney disease	1.9 (1.6-2.3)	<.01

CI, Confidence interval; COPD, chronic obstructive pulmonary disease; HR, hazard ratio.

implications. Long-term follow-up information regarding reinterventions, aneurysm-related procedures, and the cause of death are also unavailable, therefore limiting the ability to accurately assess durability of aortic interventions.

Our data set was limited to the years 2004 to 2007. Inclusion of earlier years would not have added value to our study because TEVAR was not available for widespread use before 2005. As such, patients treated before 2005 and earlier than 2004 were enrolled into ongoing FDA-approved device trials and were subject to strict inclusion and exclusion criteria that do not reflect national practices. Inclusion of patients at later dates would have been valuable in providing better definitions of practice trends and outcomes. An increased number of patients in later years would have increased the statistical power of our study, especially when analyzing outcomes by teaching status. Better analysis of trends in practice would have been possible with inclusion of later years; however, the largest changes in practice patterns noted for TEVAR and open repair occurred in the first 2 years after TEVAR became commercially available. Additional years would further define the shift of TEVAR to LV hospitals, and perhaps an increase in TEVAR cases; however, such changes are likely to be less significant than presented in this report.

Despite these limitations, the availability of a large patient cohort and the reliability of procedural and survival data contained within the MEDPAR data set has a distinct advantage over other publications using the NIS data set. Our study focused on the effect of hospital type and volume on outcomes of intact DTA repair and shows increasing use of TEVAR for DTA repair across all hospital types, with the most pronounced increase in TEVAR use at LV hospitals. Although increasing application of a new technology at inexperienced hospitals would imply increasing risk of complications and mortality for United States Medicare patients, our data suggest that this is not the case.

CONCLUSIONS

There has been a dramatic shift in DTA repair away from open repair, with increasing use of TEVAR even in LV hospitals, wherein TEVAR has eliminated the negative volume effect on perioperative outcomes seen with open surgery. These data suggest open surgical repair should be concentrated in HV centers, whereas TEVAR can be safely performed across a spectrum of hospitals.

AUTHOR CONTRIBUTIONS

Conception and design: VP, SM

Analysis and interpretation: VP, SM, EE, NA, MC, GL, CK, RC

Data collection: SM

Writing the article: VP, RC

Critical revision of the article: VP, SM, EE, NA, MC, GL, CK, RC, SM, RC

Final approval of the article: VP, SM, EE, NA, MC, GL, CK, RC

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